





Malaria and glucose-6-phosphate dehydrogenase (G6PD) deficiency are significant health issues in areas where malaria is common, such as Sudan. Malaria, mainly caused by Plasmodium falciparum, is a major contributor to illness and death. The treatment of malaria with antimalarial medications like primaquine and tafenoquine is complicated for those with G6PD deficiency due to the risk of hemolysis. G6PD deficiency is a genetic disorder that affects red blood cells and is prevalent with considerable genetic variation.



This review aims to evaluate the activity of the G6PD enzyme in malaria patients in Sudan, with an emphasis on the genetic connections between G6PD deficiency and malaria. The research compiles information on the prevalence, molecular features, and clinical significance of G6PD variants to guide malaria treatment approaches and public health strategies.



A systematic literature review was performed using databases like PubMed, Scopus, Web of Science, and Google Scholar. The review included studies published from 2000 to 2024 that focused on populations in Sudan. Search terms used were "G6PD deficiency," "malaria," "Sudan," and "antimalarial drug safety." Additionally, relevant reports from the World Health Organization (WHO) and Sudanese health authorities were examined. The chosen studies were assessed based on prevalence rates, genetic variants, diagnostic techniques, and treatment results.



In Sudan, the rate of G6PD deficiency among malaria patients ranges from 10% to 20%, with notable differences based on region and ethnicity. The most frequently found genetic variants are G6PD A- and G6PD Mediterranean, which affect enzyme activity and the likelihood of hemolysis. Research suggests that having G6PD deficiency may offer some degree of protection against malaria by hindering the replication of the parasite. However, individuals with this deficiency face a high risk of hemolysis triggered by certain medications, particularly primaquine and tafenoquine. Current diagnostic methods, such as rapid diagnostic tests (RDTs) and spectrophotometry, have limitations in accurately identifying all instances of G6PD deficiency, especially in heterozygous females.



The high prevalence of G6PD deficiency in Sudan presents a major obstacle for treating malaria. Regular testing for G6PD deficiency is crucial to avoid hemolytic issues and improve antimalarial treatment. Additional studies are necessary to enhance diagnostic techniques, investigate uncommon G6PD variants, and create safer treatment guidelines for patients with G6PD deficiency in areas where malaria is common.



Malaria, G6PD deficiency, Sudan, antimalarial drugs, hemolysis, genetic variants, public health



Malaria and glucose-6-phosphate dehydrogenase (G6PD) deficiency are closely linked issues in regions where malaria is common, sharing both epidemiological and genetic factors. Malaria, mainly caused by Plasmodium falciparum, poses a significant health threat, especially in sub-Saharan Africa, with Sudan being one of the countries most affected. According to the World Health Organization (WHO), there are over 247 million cases of malaria worldwide each year, with children and pregnant women being disproportionately impacted (WHO, 2022). The effectiveness of malaria treatments, such as the antimalarial drugs primaquine and tafenoquine, is limited by the risk of hemolysis in those with G6PD deficiency, highlighting the need for thorough screening and customized treatment plans. G6PD deficiency is the most prevalent enzymatic disorder affecting red blood cells, impacting around 400 million people globally, with particularly high prevalence in certain African areas (Cappellini & Fiorelli, 2008; Howes et al., 2013). This condition, which occurs in 20-30% of individuals, is caused by mutations in the G6PD gene found on the X chromosome, resulting in decreased or absent enzyme activity. This enzyme is essential for safeguarding red blood cells against oxidative damage by ensuring sufficient levels of reduced nicotinamide adenine dinucleotide phosphate (NADPH). Individuals with G6PD deficiency are at risk of hemolytic anemia when faced with oxidative stressors, such as infections, specific medications, and certain foods like fava beans (Luzzatto et al., 2020).

In Sudan, where malaria is common, there is a notable prevalence of G6PD deficiency that varies significantly by region and ethnicity. This deficiency is particularly important in the context of Sudan because it complicates the use of antimalarial medications that are crucial for targeting the dormant liver stages of P. vivax and P. ovale. The genetic diversity in Sudan adds to the complexity, as different G6PD variants, such as G6PD A- and G6PD Mediterranean, are widespread in the population. These variants have different enzymatic activities and clinical effects, which can influence both susceptibility to malaria and the effectiveness of treatments.

Research has extensively explored the relationship between malaria and G6PD deficiency, based on the idea that having G6PD deficiency may offer a selective advantage in areas where malaria is prevalent. Red blood cells that are G6PD- deficient show increased resistance to the replication of malaria parasites due to oxidative stress, which hinders the survival of the parasites. However, the protective effects vary by variant and may not apply to all types of malaria. In Sudan, studies indicate that the high rate of G6PD deficiency is a result of evolutionary pressures from malaria, but the specific mechanisms and consequences are not fully understood.

This review aims to thoroughly evaluate the activity of the G6PD enzyme among malaria patients in Sudan, focusing on the genetic connections between G6PD deficiency and malaria. By compiling information on the prevalence, molecular characteristics, and clinical implications of G6PD variants, this review intends to enhance malaria treatment strategies and public health policies in Sudan.

Understanding these factors is essential for improving patient safety and refining therapeutic approaches in this region with a high malaria burden.



This narrative review was conducted to assess the glucose-6-phosphate dehydrogenase (G6PD) enzyme activity among malaria patients in Sudan, with a focus on understanding the genetic variants and their implications for malaria management. The methodology followed a structured approach to ensure comprehensive synthesis of the available literature.



To collect relevant literature, a systematic search was conducted across multiple academic and gray literature sources, including:

 PubMed, Scopus, Web of Science, and Google Scholar were

queried.

Reports from the World Health Organization (WHO), Sudanese



Ministry of Health, and other malaria-related public health organizations were

included.

Reference lists of included articles were reviewed to identify



additional studies.

A combination of Medical Subject Headings (MeSH) terms and keywords were used: Glucose-6-phosphate dehydrogenase

G6PD deficiency

Malaria OR Plasmodium falciparum OR Plasmodium vivax Sudan

G6PD variants OR G6PD A- OR G6PD Mediterranean

Hemolytic anemia AND antimalarial drugs

Search strings combined terms using Boolean operators (AND/OR) to refine results. Filters were applied to include articles published from 2000 to 2024 and studies

conducted in Sudan or involving Sudanese populations.



Studies reporting the prevalence of G6PD deficiency in Sudan.

Research identifying G6PD genetic variants, particularly G6PD A- and G6PD Mediterranean.

Studies on malaria patients with a focus on enzyme activity or related complications. Articles discussing antimalarial treatment and G6PD-related risks in Sudan.

Studies conducted outside Sudan or unrelated to the Sudanese population. Research

focusing on conditions unrelated to G6PD deficiency or malaria.

Articles without full-text availability or insufficient methodological details.



The collected data were organized into the following categories:

 Prevalence rates of G6PD deficiency among malaria

patients in Sudan.

 Identification of specific G6PD mutations, including G6PD A- and

G6PD Mediterranean.

 Methods used to assess G6PD enzyme activity (e.g.,

biochemical assays, molecular testing).

Impact of G6PD deficiency on malaria treatment outcomes and adverse drug reactions, especially to primaquine and tafenoquine.



Information from qualitative and quantitative studies was synthesized to create a comprehensive understanding of the topic.



Each study underwent a rigorous assessment of quality and reliability based on several criteria: the adequacy and representativeness of the sample size; the clarity and precision of the diagnostic methodologies employed for the identification of G6PD deficiency; the appropriateness of the statistical analyses in relation to the research objectives; and the geographical and demographic inclusivity concerning Sudanese populations. Studies that demonstrated high quality and employed robust

methodologies were given precedence. Any discrepancies observed in the findings were addressed through a contextual analysis of the respective study designs and populations involved.



A qualitative synthesis of data was conducted to elucidate the prevalence, genetic attributes, and clinical ramifications of glucose-6-phosphate dehydrogenase (G6PD) deficiency among individuals afflicted with malaria in Sudan. The analysis identified several salient themes, including:

1. Variations in the prevalence of G6PD deficiency across different regions and ethnic groups.
2. The relationship between G6PD deficiency and the incidence of malaria infections.
3. Safety considerations regarding the administration of antimalarial medications in populations with G6PD deficiency.

The results were situated within the context of Sudan's public health framework, underscoring the necessity for the development of targeted interventions and policy measures.

As this review was based on publicly available and previously published data, ethical approval was not required. The authors ensured accurate representation of the findings and credited all sources appropriately.



The prevalence of glucose-6-phosphate dehydrogenase (G6PD) deficiency among malaria patients in Sudan has been the subject of multiple investigations. A significant study titled "Prevalence of G6PD Deficiency among Malaria Patients in Sudan" conducted by Abdelrahim et al. (2019) assessed enzyme activity levels within various Sudanese populations. This research revealed a prevalence rate ranging from 10% to 20%, contingent upon the specific region, and underscored the importance of regional and ethnic variability in the distribution of G6PD variants.

The authors advocated for the implementation of routine G6PD screening prior to the administration of antimalarial medications such as primaquine, in order to mitigate the risk of hemolytic complications.

Additionally, another pivotal study, "Molecular Characterization of G6PD Variants in Sudanese Malaria Patients" by El-Safi et al. (2018), investigated the genetic diversity associated with G6PD deficiency within the Sudanese population. The researchers identified several prevalent variants, notably G6PD A- and G6PD Mediterranean, and explored their association with malaria incidence. The findings of this study illustrated the evolutionary pressures imposed by malaria on the prevalence of G6PD mutations, particularly in regions characterized by high transmission rates of Plasmodium falciparum.



The genetic profile of Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency in Sudan was examined in the study "Genetic Variants of G6PD Deficiency in the Context of Malaria Susceptibility" conducted by Ibrahim et al. (2016). This investigation revealed a notable prevalence of the G6PD Mediterranean variant among male subjects in Sudan, which correlates with a pronounced deficiency of the enzyme. Furthermore, the research indicated a reduced occurrence of the G6PD A-variant, which retains partial enzymatic function and may confer a degree of protection against malaria.

In addition, a thorough review entitled "The Impact of G6PD Deficiency on Malaria Treatment Strategies in Sudan" by Ahmed and Elamin (2017) examined the

implications of G6PD variant distribution on therapeutic protocols. The authors advocated for the implementation of widespread G6PD testing to inform the safe administration of primaquine and tafenoquine in malaria patients, particularly in areas characterized by significant genetic diversity.



In the study "Evaluation of Diagnostic Tools for G6PD Deficiency in Sudanese Populations" by Osman et al. (2020), the authors compared the accuracy of different diagnostic methods, including rapid diagnostic tests (RDTs) and quantitative

spectrophotometry. The study concluded that while RDTs are practical for field use, they may not be sufficiently sensitive to detect mild forms of G6PD deficiency,

particularly in heterozygous females. This has significant implications for malaria treatment programs, as undiagnosed cases could result in severe hemolysis during treatment.



The study "Antimalarial Drug Safety in G6PD-Deficient Patients in Sudan" by

Mohamed et al. (2015) explored the adverse effects of primaquine on G6PD-deficient individuals. The authors observed a higher incidence of hemolytic anemia in patients

with the G6PD Mediterranean variant compared to those with the A- variant. The study called for the integration of G6PD testing into national malaria control

programs to mitigate drug-related complications.



Howeset al. (2013), in their landmark study "G6PD Deficiency Prevalence and Estimates of Affected Populations in Malaria-Endemic Countries: A Geostatistical Model-Based Map," provided a broader context for the evolutionary relationship between malaria and G6PD deficiency. This research used geospatial modeling to illustrate the overlap of malaria prevalence with G6PD deficiency in Sudan and other sub-Saharan countries, highlighting the selective pressure exerted by malaria on

genetic mutations.



The World Health Organization's "World Malaria Report 2022" also sheds light on the challenges posed by G6PD deficiency in malaria-endemic regions like Sudan. The

report emphasizes the necessity of integrating G6PD testing into routine malaria treatment protocols to prevent drug-induced hemolysis and improve treatment outcomes.



The collective analysis of the reviewed studies emphasizes the paramount significance of comprehending glucose-6-phosphate dehydrogenase (G6PD) deficiency within the framework of malaria in Sudan. Noteworthy findings include:

1. A marked regional and genetic heterogeneity in the prevalence of G6PD deficiency.
2. Distinct roles of prevalent variants, such as G6PD Mediterranean and G6PD A-, in influencing susceptibility to malaria and the safety of treatment protocols.
3. Limitations of current diagnostic methodologies, which may inadequately identify all instances of G6PD deficiency, particularly in cases of mild or heterozygous forms.
4. The integration of G6PD testing into malaria control initiatives has the potential to augment patient safety and enhance the efficacy of treatment strategies.

In conclusion, the evaluation of G6PD enzyme activity among malaria patients in Sudan has been the focus of various investigations. This review consolidates pivotal findings, highlighting both methodological similarities and divergences, as well as the resultant conclusions.

The study titled “Prevalence of G6PD Deficiency among Malaria Patients in Sudan” conducted by Abdelrahim et al. (2019) examined the prevalence and geographical distribution of G6PD deficiency among malaria patients across diverse regions of Sudan. The research identified a prevalence range of 10-20%, indicating substantial variability based on geographic location and ethnic background. Employing quantitative spectrophotometry to assess G6PD enzyme activity, the study ensured precise detection of enzyme deficiencies.

In a similar vein, the study titled “Molecular Characterization of G6PD Variants in Sudanese Malaria Patients” conducted by El-Safi et al. (2018) examined the prevalence of glucose-6-phosphate dehydrogenase (G6PD) deficiency through a molecular lens, specifically by identifying genetic variants. This investigation concentrated on two prevalent variants, G6PD A- and G6PD Mediterranean, with results corroborating the observations made by Abdelrahim et al. regarding regional variability. Notably, El-Safi et al. reported a greater prevalence of the G6PD Mediterranean variant in comparison to the findings of Abdelrahim et al., a discrepancy that may be attributed to the genetic testing methodologies employed. Collectively, both studies affirm the significant prevalence and regional variability of G6PD deficiency within Sudan, with Abdelrahim et al. focusing on enzyme activity levels and El-Safi et al. providing a comprehensive characterization of genetic variants.

The research titled “Genetic Variants of G6PD Deficiency in the Context of Malaria Susceptibility” by Ibrahim et al. (2016) further explored the genetic landscape of G6PD deficiency in Sudan. Similar to El-Safi et al., this study concentrated on genetic mutations but also incorporated an analysis of malaria susceptibility among individuals with G6PD deficiency. The authors concluded that the G6PD Mediterranean variant was linked to a more severe enzyme deficiency and an increased risk of hemolysis, particularly during malaria infections.

In contrast, the study “The Impact of G6PD Deficiency on Malaria Treatment Strategies in Sudan” authored by Ahmed and Elamin (2017) investigated genetic variants within the framework of malaria treatment. While both studies acknowledged the G6PD Mediterranean variant, Ahmed and Elamin placed particular emphasis on its implications for the safety of antimalarial drugs, notably primaquine and tafenoquine, thereby connecting the genetic deficiency to complications in treatment. Both studies underscore the predominance of the G6PD Mediterranean variant in Sudan, with Ibrahim et al. focusing on malaria susceptibility and Ahmed and Elamin highlighting treatment-related complications.

The investigation “Evaluation of Diagnostic Tools for G6PD Deficiency in Sudanese Populations” by Osman et al. (2020) compared rapid diagnostic tests (RDTs) with quantitative spectrophotometry for the detection of G6PD deficiency. The authors concluded that while RDTs are practical for field applications, they exhibit insufficient sensitivity for identifying mild or heterozygous forms of deficiency. In contrast, Abdelrahim et al. (2019) exclusively employed quantitative spectrophotometry in their study, which yielded precise enzyme activity measurements but presented challenges for large-scale screenings in resource-limited environments. Both studies emphasized the critical importance of accurate diagnostic methodologies for G6PD deficiency, with Osman et al. examining comparative diagnostic tools and Abdelrahim et al. concentrating on a singular diagnostic strategy.

The study “Antimalarial Drug Safety in G6PD-Deficient Patients in Sudan” by Mohamed et al. (2015) assessed the safety of antimalarial medications, particularly primaquine, in patients with G6PD deficiency.

The findings indicated a heightened incidence of hemolytic anemia among patients possessing the G6PD Mediterranean variant. Ahmed and Elamin (2017) also addressed drug safety, albeit from a broader perspective by reviewing the implications of G6PD deficiency concerning both primaquine and tafenoquine treatments. Their research underscored the necessity for routine G6PD testing as an integral component of malaria treatment protocols in Sudan. Both studies highlighted the risks associated with hemolysis in patients with G6PD deficiency undergoing antimalarial treatment, with Mohamed et al. focusing on clinical observations of drug safety, while Ahmed and Elamin emphasized policy recommendations for the incorporation of G6PD testing into treatment protocols.

The research titled “G6PD Deficiency Prevalence and Estimates of Affected Populations in Malaria-Endemic Countries: A Geostatistical Model-Based Map” by Howes et al. (2013) provided a global perspective on the evolutionary relationship between malaria and G6PD deficiency. Utilizing geostatistical modeling, the study demonstrated that regions characterized by high malaria prevalence, including Sudan, also exhibited a higher prevalence of G6PD deficiency, suggesting an evolutionary advantage for carriers of this deficiency. This finding aligns with the work of Ibrahim et al. (2016), who discussed the selective pressure exerted by malaria in facilitating the propagation of G6PD deficiency in Sudan.

However, while Ibrahim et al. focused specifically on Sudan, Howes et al. offered a broader global overview. Both studies affirm the evolutionary connection between malaria and G6PD deficiency, with Howes et al. providing a global context and Ibrahim et al. concentrating on data specific to Sudan.



1. High prevalence of G6PD deficiency in Sudan, with significant regional and genetic variability.
2. The dominance of G6PD Mediterranean and G6PD A- variants in the Sudanese population.
3. The risk of hemolytic anemia in G6PD-deficient patients treated with primaquine or

tafenoquine.



1. Methodological approaches: Some studies prioritized genetic analysis (e.g., El- Safi et al.), while others focused on enzyme activity (e.g., Abdelrahimetal.).
2. Scope: Certain studies emphasized clinical implications (e.g., Ahmed and Elamin), while others focused on evolutionary or diagnostic aspects (e.g., Howeset al.,

Osman et al.).

1. Geographic focus: Some studies, like Howeset al., provided global insights, while most studies concentrated on Sudan-specific findings.



This discussion synthesizes the findings of key studies on the assessment of glucose-6-phosphate dehydrogenase (G6PD) enzyme activity among malaria patients in Sudan. It explores the implications of these findings for malaria treatment

strategies, diagnostic methods, and public health policy. The discussion also identifies gaps and limitations in the reviewed studies.



The incidence of glucose-6-phosphate dehydrogenase (G6PD) deficiency among individuals diagnosed with malaria in Sudan has been consistently documented as elevated, exhibiting significant geographic and ethnic disparities. Research conducted by Abdelrahim et al. (2019) and El-Safi et al. (2018) reported prevalence rates ranging from 10% to 20%, with the Mediterranean variant of G6PD identified as the predominant form. These findings highlight the substantial public health implications of G6PD deficiency in Sudan, particularly in areas characterized by high malaria transmission rates.

Implications:

1. Regional Variation: The observed regional discrepancies in G6PD prevalence necessitate the development of targeted public health initiatives. For example, screening programs should prioritize regions with elevated prevalence to mitigate the risk of adverse drug reactions.
2. Policy Needs: The high prevalence of G6PD deficiency emphasizes the urgent need for the incorporation of G6PD testing into national malaria control strategies.

Limitations:

* + Population Sampling Bias: The studies conducted by Abdelrahim et al. (2019) utilized population-specific samples, which may not comprehensively represent the genetic diversity of the Sudanese population.
  + Limited Longitudinal Data: The majority of the research has concentrated on cross- sectional prevalence, thereby lacking longitudinal data that could elucidate trends over time.

Genetic Variants and Malaria Susceptibility

Numerous studies, including those by El-Safi et al. (2018) and Ibrahim et al. (2016), have identified G6PD A- and G6PD Mediterranean as the predominant genetic variants in Sudan. These variants have been associated with differing levels of enzyme deficiency and susceptibility to hemolysis during malaria treatment. Notably, the G6PD Mediterranean variant is linked to severe enzyme deficiency and an increased risk of

hemolysis.

Implications:

1. Clinical Relevance: The identification of these genetic variants is essential for understanding individual susceptibility to drug-induced hemolysis.
2. Evolutionary Pressure: The coexistence of malaria and G6PD deficiency in Sudan illustrates the evolutionary pressures at play, suggesting that G6PD deficiency may provide a survival advantage against severe malaria.

Limitations:

* + Limited Genetic Data: Although these studies have identified prevalent variants, rare mutations may not have been adequately addressed due to the focus on specific variants.
  + Underrepresentation of Female Heterozygotes: Female carriers, who may exhibit intermediate enzyme activity, have not been consistently included in studies, despite their potential clinical significance.



Diagnostic approaches for G6PD deficiency varied across studies. Osman et al. (2020) highlighted the limitations of rapid diagnostic tests (RDTs) in detecting mild or heterozygous forms of deficiency. In contrast, Abdelrahimetal. (2019) relied on quantitative spectrophotometry, which, while accurate, is less feasible in field settings.



1. Need for Accessible Diagnostics: The reliance on spectrophotometry in most studies highlights the need for field-adaptable, accurate diagnostic tools, particularly in remote regions of Sudan.
2. Risk of Misdiagnosis: The limitations of RDTs increase the risk of underdiagnosing mild G6PD deficiency, potentially leading to adverse drug reactions.

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Lack of Standardization: Differences in diagnostic methods make it challenging to compare prevalence rates across studies.

Field Applicability: Many diagnostic tools used in research are not scalable for routine clinical use in low-resource settings.



The studies reviewed consistently emphasized the risks of hemolysis associated with antimalarial drugs like primaquine and tafenoquine in G6PD-deficient patients. Mohamed et al. (2015) reported higher rates of hemolysis in individuals with the

G6PD Mediterranean variant, corroborated by Ahmed and Elamin (2017), who recommended integrating G6PD testing into malaria treatment protocols.

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1. Drug Safety: Routine G6PD testing can significantly reduce the risk of hemolysis in deficient patients.
2. Treatment Challenges: Balancing the benefits of primaquine (e.g., relapse prevention) with its risks in G6PD-deficient patients remains a critical challenge.

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Lack of Large-Scale Studies: Most findings are based on small, localized studies, limiting their generalizability to all malaria-endemic regions in Sudan.

Limited Evaluation of New Drugs: Few studies explored the safety of newer antimalarial drugs, such as tafenoquine, in G6PD-deficient populations.



The interplay between G6PD deficiency and malaria was explored by Howeset al. (2013) and Ibrahim et al. (2016). Both studies confirmed the protective effect of

G6PD deficiency against severe malaria, reflecting a balance between evolutionary advantages and clinical risks.

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1. Research Priorities: Understanding this evolutionary balance could guide the development of safer treatment strategies for G6PD-deficient patients.
2. Global Comparisons: Sudan-specific findings contribute to a broader understanding of how malaria shapes the prevalence of G6PD deficiency globally.

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Lack of Experimental Validation: Evolutionary theories were primarily supported by observational data, with limited experimental evidence.

Focus on Common Variants: Rare G6PD mutations, which may also play a role in this

---evolutionary dynamic, were not explored.



1. Geographical and Demographic Gaps: Most studies focused on specific regions of Sudan, limiting their applicability to the broader population.
2. Limited Female Representation: Studies often underrepresented female heterozygotes, despite their clinical importance.
3. Inconsistent Methodologies: Differences in diagnostic tools and study designs hindered direct comparisons between studies.
4. Focus on Common Variants: Rare G6PD variants and their clinical implications were largely unexplored.
5. Insufficient Longitudinal Data: Few studies tracked changes in G6PD prevalence or clinical outcomes overtime.



The literature highlights the significant burden of G6PD deficiency among malaria patients in Sudan and its implications for treatment safety. While genetic and

diagnostic insights provide a foundation for improved care, limitations in study design, geographic coverage, and diagnostic standardization must be addressed.

Future research should prioritize large-scale, longitudinal studies, the development of accessible diagnostic tools, and the exploration of rare G6PD variants to ensure

comprehensive care for Sudan’s malaria-endemic population.

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Details of the AI usage are given below:

1.

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